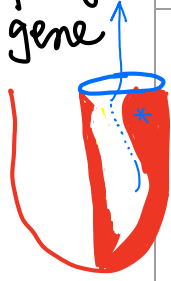


ch14 #

Cardiomyopathy clinical details

AD MYH7 gene  
β myosin ++  
gene



	<b>Hypertrophic</b> 20-40 yf: ♂ CHEST PAIN on EXERTION SYNCOPEAL ATTACKS,	<b>Dilated</b> Alcohol Sarcoidosis, DMD
<b>Histology</b>	Banana shaped cavity Sub-valvular aortic stenosis Systolic anterior movement of anterior valve Predominant diastolic malfunction Coronary blood flow ↓	Thinned out myocardium * global Hypokinesia
<b>Pulse</b>	Pulsus Bisfuriens	Dicrotic Pulse - pedal edeme - pulm edeme
<b>Murmur</b>	* Ejection systolic murmur <u>except</u> louder $\bar{c}$ Valsalva, standing, amyl nitrate	(FUNCTIONAL) MR/TR panoptotic murmur
<b>Treatment of choice</b>	→ I.C.D ± alcohol based septal ablation PROPRANOLOL + DICOPYRAMIDE	Cardiac Transplant - CRT-D warfarin + spironolactone ± digoxin

① => All MURMURS ↓  $\bar{c}$  Valsalva, standing, amyl nitrate

Restrictive cardiomyopathy RAREST, amyloidosis, Radiation, Sarcoidosis  
M/C

<b>Histology</b> STIFF VENTRICLE	gross: Atrial size > ventricle size m/c: MASSON TRICHROME: +++ blue in myocardium (collagen)
<b>Pulse</b> RV compliance ↓ LV " ↓	PULSUS PARADOXUS ± Pedal edeme, Ascites (abdo distention) SOB, ORTHOPNEA, EFFORT INTOLERANCE KUSSMAUL SIGN
<b>JVP finding</b>	 Pericardial shudder
<b>IOC</b>	Cardiac MRI vs endomyocardial Biopsy gold std ↓
<b>Cardiac catheterization</b>	Square root wave sign ±

Rx: Cardiac Tx, spironolactone, warfarin

death of loved one!

**Takotsubo cardiomyopathy/ Broken heart syndrome**

- Mimics MI presentation
- Elevated cardiac biomarkers with ST segment elevation
- Diagnosis made in cath lab Coronary angiography

TRAPPED in an elevator during earthquake

Catecholamines + SURGE

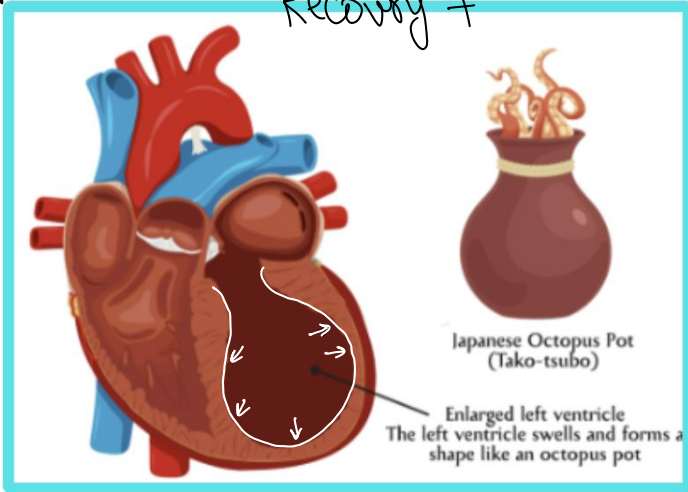
Chest pain at Rest  
ST ↑  
Troponin I elevated  
T  
CK-MB

dobutamine  
Nor epinephrine  
☹️

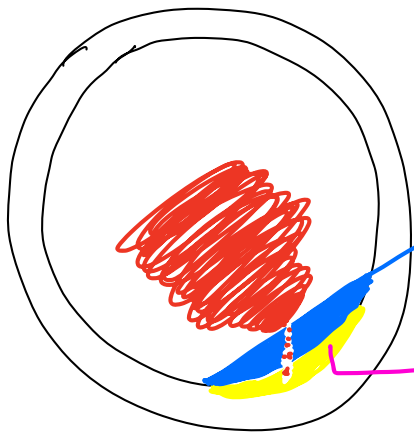
Management: I.A.B.P

**NORMAL**

Recovery +



MCC death in india  
↳ I.H.D



FIBROUS CAP

Foam cells  
Macrophages + oxidized LDL

Fatty STREAKS

PLAQUE FISSURE

Collagen FISSURE

platelet aggregation: THROMBUS

MC BV involved in atherosclerosis  
↓  
Abdominal AORTA



#  
LAD → LV: Ant wall: V2 V3 V4  
RCA → RV: inf wall: **lead II, III, aVF**  
#

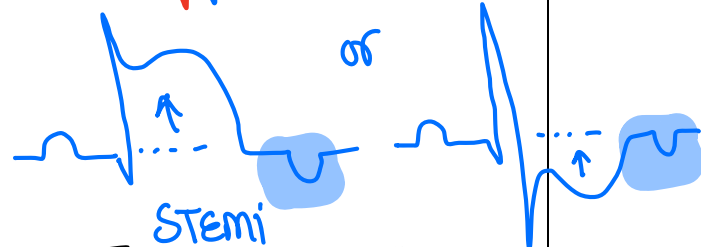
\* EXCRUCIATING CHEST PAIN, Radiation of pain Arm (L), (L) shoulder, jaw, epigastrum

Myocardial infarction

✓ Earliest ECG finding  
**HYPERACUTE T wave**



✓ ECG finding that persists for whole life/ Old MI  
**deep Q wave**



First cardiac biomarker to rise in MI  
 Ischemia modified albumin: < 1 HOUR  
 HEART fatty acid binding protein: 1-3 HOUR  
 Troponin I/T = 3-6 HOURS  
 Last to rise in MI = **LDH** (BEST)

**STEMI**  
 Full thickness #  
 FIBRIN Rich  
 Clot  
 FIBRINOLYSIS 😊

**NSTEMI**  
 partial #  
 Thickness  
 Platelet Rich  
 Clot

Cardiac biomarker of reinfarction > 72 hours : CPK-MB  
 " " " " : Troponin I: ↑ 20% elevation over (admission value)

Treatment of Choice for STEMI  
 ↳ \* **Primary percutaneous coronary intervention**  
 NSTEMI → PCI

iv TIROFIBAN  
 iv EPTIFIBATIDE  
 iv CANGREOR  
 → gp IIb/IIIa ⊖

First medical contact to balloon time  
 ↳ < 90 minutes

Window period for thrombolysis: < **12** HRS of **symptom onset**  
 = 30/90 | 6 hr | 12 HOUR  
 min min

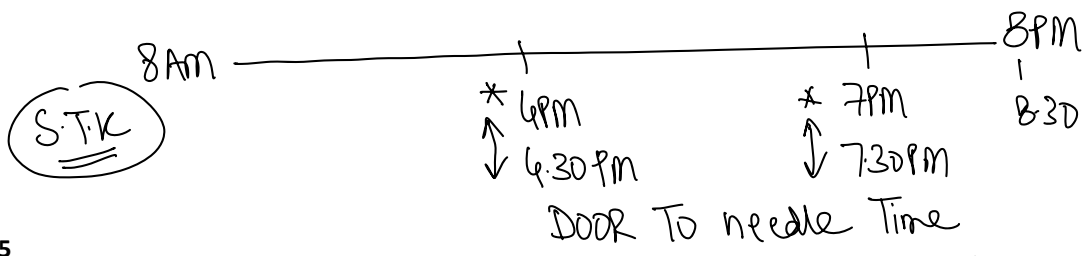
Door to needle time (cath lab facility is not available)  
 ↳ < 30 minutes

Rescue PCI (< 50% ST-segment resolution at 60-90 min after fibrinolysis)

↳ failure of Thrombolysis



**GOLDEN PERIOD MI = 1-3 HRS**



## Complications of myocardial infarction

Most common complication in few hours: **V-FIB, V-Tach**

MC cause of death > 24 hours → **CARDIOGENIC SHOCK**

Death 3-5 days → **ELECTROMECHANICAL DISSOCIATION**

MC valvular lesion seen in MI → **M.R**

Dressler syndrome → **Aspirin**  
autoimmune pericarditis

Cardiac Rupture

**AIVR: REPERFUSION Arrhythmia**  
(slow V-T)



### Mnemonic

**C:** Cardiogenic shock

**A:** Arrhythmias

**L:** LV aneurysm

**M:** Mitral regurgitation (papillary muscle dysfunction / rupture)

**D:** Dressler



Rx: **BEST-MOAN**

Beta blockers - Enoxaparin - statins - **THROMBOLYSIS**  
**TIROFAN**

Morphine - O<sub>2</sub> - Aspirin - NTG  
Atorvastatin, ACEc

1. Aspirin chewable + Ticagrelor: **DAPT**

2. O<sub>2</sub> SpO<sub>2</sub> < 93%: **HUDSON MASK**

3. SL NTG CI: SBP < 90/60

4. Morphine: vagotonic action: **CONSERVE FUEL/O<sub>2</sub>**

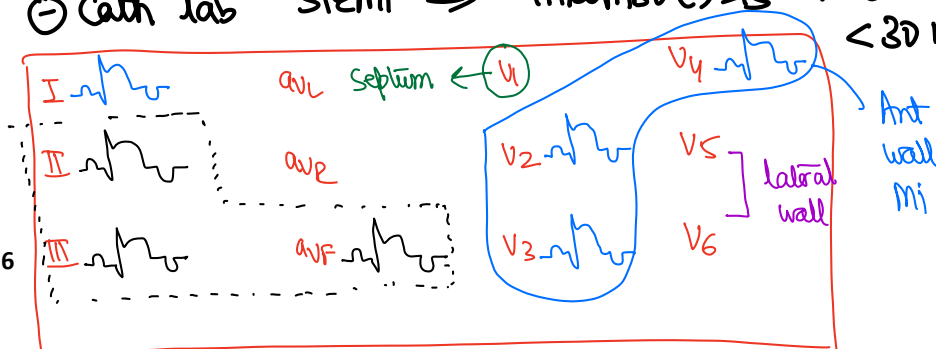
5. Metoprolol CI: SBP < 90/60, vly wall MI

6. Enoxaparin: ⊖ propagation of clot

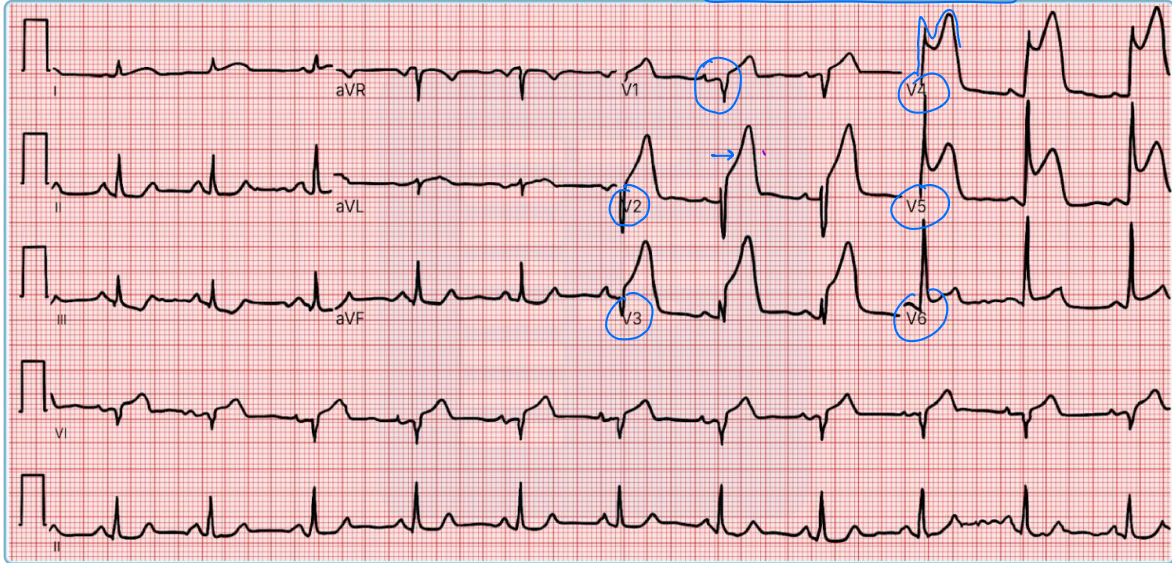
7. **ATORVASTATIN, ACEc**

8. Cath lab: STEMI ⇒ Primary P.C.I: FMC - balloon

⊖ Cath lab STEMI ⇒ **THROMBOLYSIS** FMC - needle < 30 min

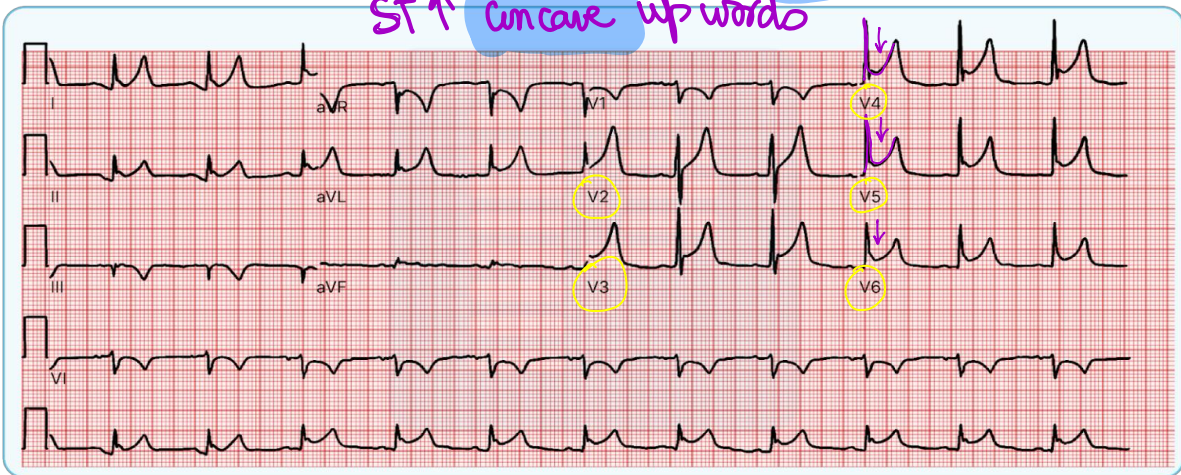


EXTENSIVE Ant wall MI



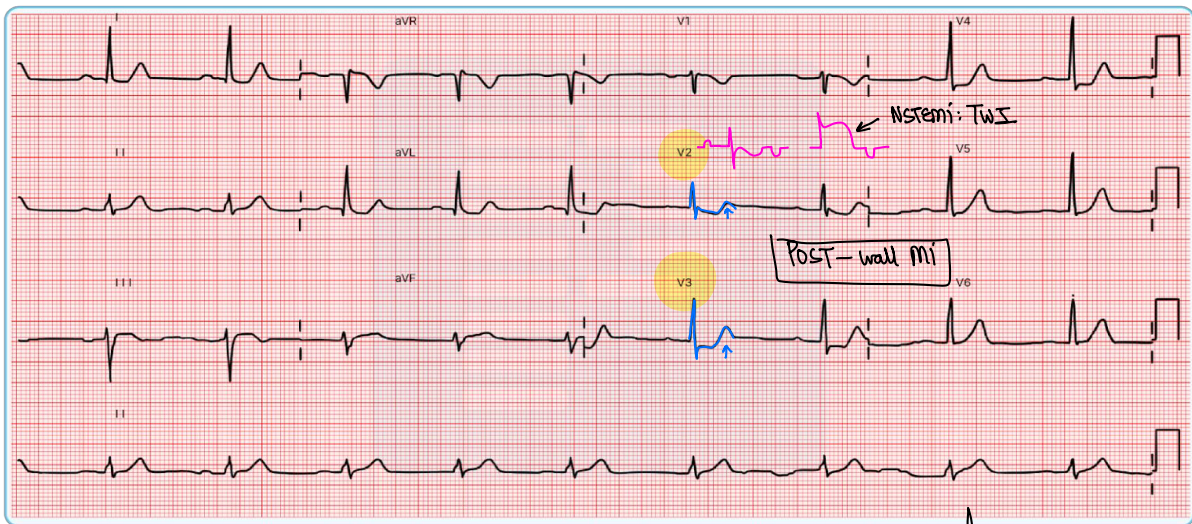
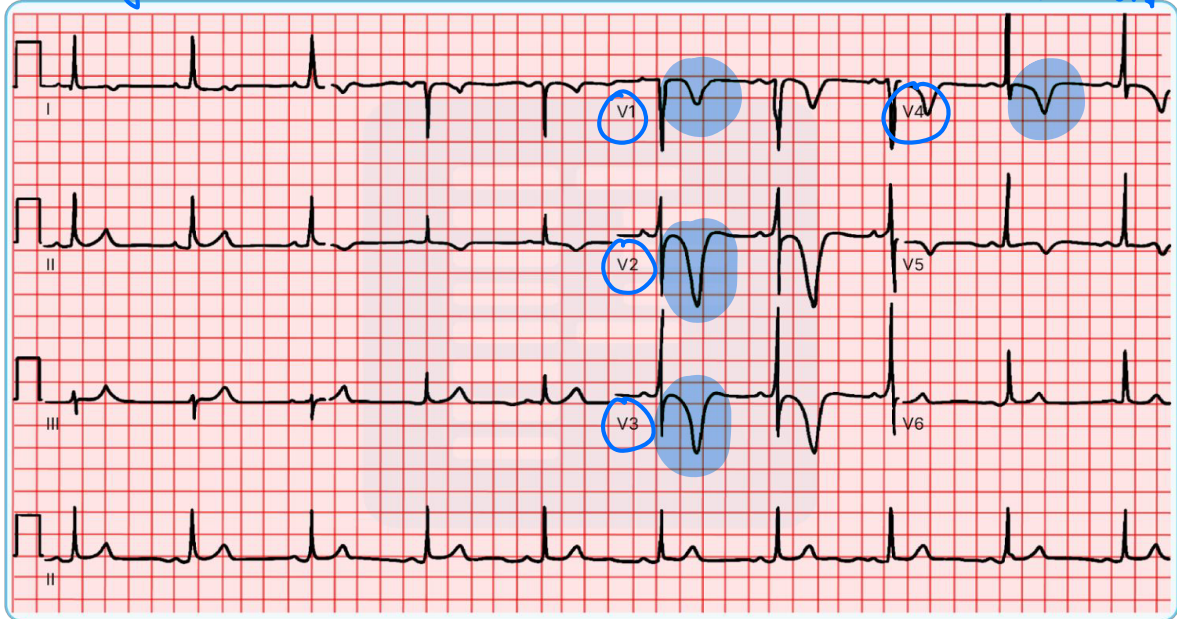
### Acute PERICARDITIC


POST-URTI, do CHEST PAIN Rest x 3 days  
ST ↑ concave up words



STATIN/ Aspirin/ STK/ NTG  
 Not given in this case?

NSTEMI: T wave inversion



\* POST-WALL MI  $\Rightarrow$   $\frac{V_2, V_3, V_4}{V_7, V_8, V_9} \Rightarrow$  





## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes



## Additional Notes

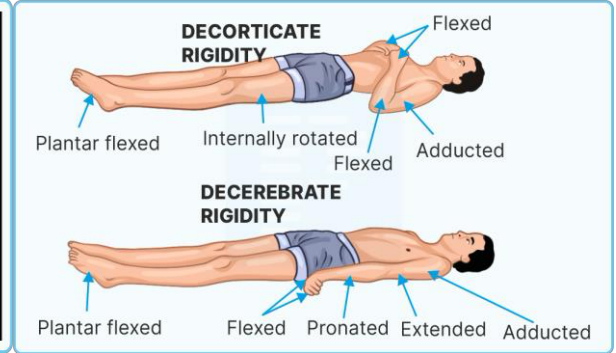
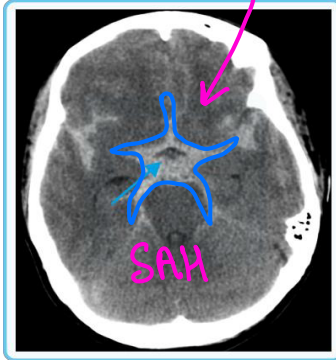


## Additional Notes

STAR of death

lined gland calcification

Neurology



Chroid plexus Calcification

**Seizures and epilepsy**

MCC of acquired epilepsy in India is : NCC : Intra cranial calcification

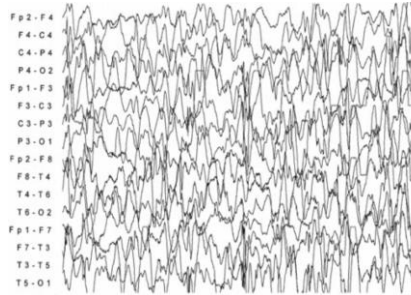
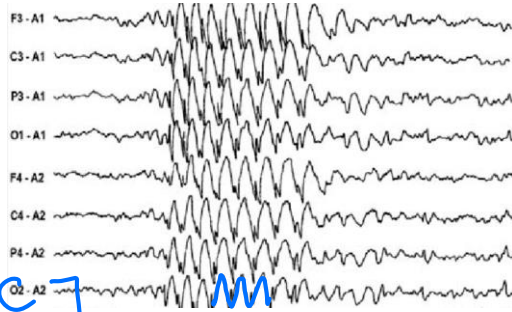
Normal EEG finding with eyes open:

$\beta$  : 13-30Hz 1

Normal EEG findings with eyes closed:

$\alpha$  : 8-13Hz 0

EEG finding of < 3 Hz spike and slow wave pattern and multiple seizures types is seen in **LGS**



Ethosuximide/ Valproate/ Lamotrigine

ACTH / Vigabatrin if associated with TS

Scholastic performance poor \* **HYPERTHIMIA**

Vacant staring spells

3/sec spike & slow

ABSENCE / petit Mal epilepsy

\* PHENYTOIN, CBZ: ☹️

WORSENS ↑

\* CBZ ⇒ SIADH — Na ↓

TLC: agranulocytosis

# GCSE : Status epilepticus

Drug of choice for Seizures in patient >5 minutes in a patient with GTCS

No Touch policy

1. iv Lorazepam and repeat iv LORAZEPAM if necessary or intranasal midazolam / im lorazepam
2. Start Phenytoin or Valproate or Levetiracetam infusion

If failure to respond to BZD 2 doses and 1<sup>st</sup> line AED then diagnose as Refractory SE

3. Propofol : Rapid sequence intubation, ET

T1 = time to treat and T2 = risk of neuronal injury  
~5min ~30min

green urine in vrobag

Focal seizures

Starts with Aura : visual blurring

Jacksonian march or Marching epilepsy epilepsy with Todd paralysis

↳ inability to use arm, leg: variable time

Complex hallucinations and automatisms.

Burning RUBBER / DEAD FISH

↳ involuntary smack lip like

MRI shows hippocampal sclerosis. Diagnosis:

±

=

↳ epileptogenic focus

Mesial Temporal lobe sclerosis

Rx : Neuro-surgery

CBZ

LCOP ⇒

LAMOTRIGINE, CBZ, OXCARBAZEPINE

PHENYTOIN

# \* PREMONITORY SYMPTOMS



<p><b>GTCS</b></p> <p><u>M/C</u></p> <p>Valproate levetiracetam or lamotrigine</p> <p>♀</p>	<p>Ictal cry → <u>TONIC</u> → <u>CLONUS</u></p> <p>Usually treatment is given for 2 years and then tapering is done</p> <p>Lamotrigine can be used in <u>both</u> GTCS and focal seizures</p> <p>Clonus: Violent JERKING all limbs uprolling eyeballs Frothing corners <u>PERIORAL CYANOSIS</u> Tongue bites +/-</p> <p>15-30 sec</p>
<p><b>Absence seizures</b></p>	<p>Sudden brief loss of consciousness <u>without</u> loss of postural control</p> <p>Vacant <u>staring</u> spells <u>with</u> → <u>ATONIC</u> s/drop attacks</p> <p>EEG finding of <u>3/sec</u> spike and slow wave <u>Rx: Valproate</u></p> <p>Carbamazepine and phenytoin worsen absence seizures</p> <p>good prognosis: age ↑ severity ↓</p>

- \* Valproate ⇒ HYPERAMMONEMIA (LFT), alopecia, wt loss
- MC type of epilepsy in children: → Benign Rolandic PANCREATIC
- MC type of seizures in children: → Febrile seizures epilepsy ↓ \* focal seizures
- MC type of seizures in neonates: → Subtle seizures do JITTERINESS
- Leading cause of subtle seizures: H.I.E

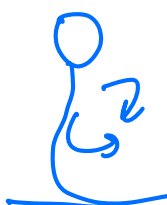
## ✓ Febrile seizures

Best drug for intermittent prophylaxis: → ORAL CLOBAZAM / Diazepam

\* Best drug for continuous prophylaxis: oral valproate Don't use phenytoin

✓ Best drug for management of acute episode at home: → Rectal diazepam  
intranasal midazolam

8mth



Salaam seizure:



myoclonic JERK: Trunk

EEG: grossly chaotic rhythm

inj ACTH

E.coli: (india)

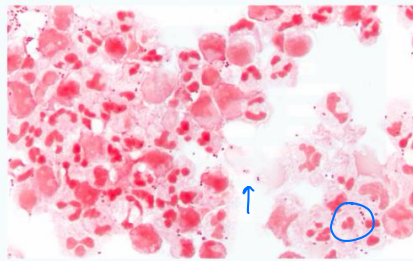
Overtaking Reaction India Link

Septic SHOCK: NS + NORAD  
TARGET MAP: > 65 mm Hg

**Pyogenic Meningitis**

Strep pneumoniae

gm ⊖ bc: N.meningitidis



MCC in neonate with bulging AF and vacant stare: *Strep. agalactiae* > *Listeria*  
Rx: CEFOTAXIME + ampicillin ..... -> monocytes

Leading cause of pyogenic meningitis in adults > 3 months to < 55 years

*Strep. pneumoniae* adult / child

Leading cause of pyogenic meningitis in adults with purpuric rash and distributive shock

*N.meningitidis*: Dic: petechiae, purpura

Perform guarded LP and then start Empirical antibiotic treatment within 60 minutes with

\* CEFTRIAXONE + VANCOMYCIN

Add coverage for + AMPICILLIN (Listeria) in alcoholics/ heart transplant

\* Mass Chemoprophylaxis for N.meningitidis with CIPROFLOXACIN  
HCW " " " " -> CEFTRIAXONE

Recurrent meningitis in adults with meningococcus is due to C5-C9 deficiency -> Meningococcus

**Whether neuroimaging or LP first in case of meningitis**

Always do neuroimaging first if any one present

MAC

- ✓ Papilledema
- ✓ Immunocompromised state
- ✓ New-onset seizures
- ✓ Focal neurological deficit
- ✓ Altered sensorium / low GCS
- ✓ Neoplasm / known CNS lesion

PIN-FAN  
any 1 +  
PERFORM => neuroimaging

- \* FEVER + HEADACHE + nuchal rigidity
- ✓ rule out Raised ICP: fundoscopy / NCCT head
- ✓ guarded LP: mfc: PMN +++
- ✓ iv: send blood culture -> start Antibiotics

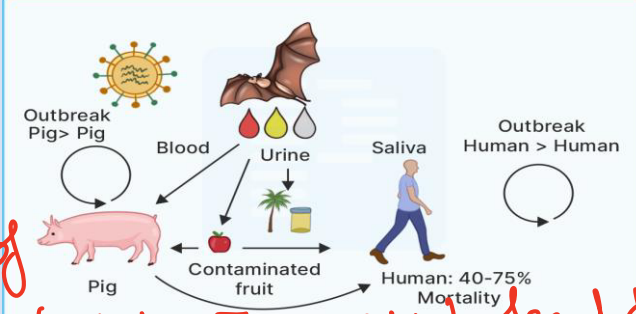
Viral encephalitis

Hippocampus #

TEMPORAL

<p>Overall MCC</p> <p>↓</p> <p>HSV-1</p> <p>FEVER x 1 day</p> <p>ALTERED sensorium</p> <p>O/E: ⊖ nuchal rigidity</p>	<ul style="list-style-type: none"> <li>- Shows predilection for <u>TEMPORAL</u> lobe</li> <li>* - CSF shows RBC in CSF</li> <li>* - EEG Shows PLED <i>Periodic lateralized epileptiform discharge</i></li> <li>- Empirical Rx started with <u>Acyclovir</u></li> <li>- MRI heads: Temporal lobe inflammation</li> <li>IOC: CSF PCR HSV-1</li> </ul>
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<p>Leading cause in India</p> <p>U.P. (GORAKHPUR)</p>	<p>Japanese B encephalitis</p> <p>(COLEX Mosquito)</p> <p>Rx = ? 1<sup>o</sup>: vaccine</p>
---	---

<p>Kerala outbreak</p> <p>SILIGURI</p> <p><u>Nipah virus</u></p> <p>* Consumption of fruits contaminated w/ saliva fecal droppings of fruit BAT ⇒ <u>PTEROPUS</u></p> <p>* DATE PALM SAP</p> <p>* CFR = 40-75%</p>	 <p>Human: 40-75% Mortality</p> <p>death: encephalitis (microhemorrhages in brain)</p>
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\* Viral meningitis ⇒ ENTEROVIRUS: Coxsackie B

FEVER + headache + nuchal rigidity

U: CSF: lymphocytes ++, sugar: Normal

protein ↑

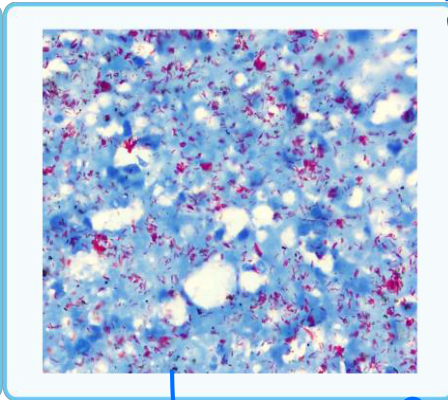
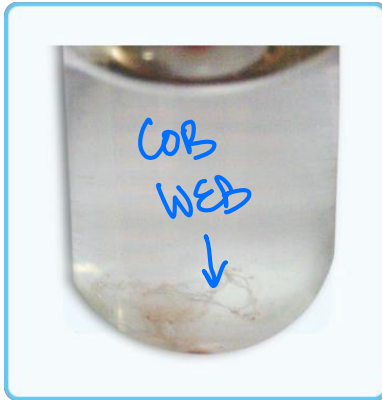
\* LP = CI Raised ICP, Risk of Tonsillar Herniation



Findings of CSF

Remarks	Cells	Sugar	Protein	Opening pressure	Color
Normal	0-5 lymphocytes	$\frac{2}{3}$ (bid sups)	15-45 mg/dL	10-20 mmHg dex 50-180 mm H <sub>2</sub> O	
ABM Pyogenic Rx → CEFTRIAXONE + vancomycin Antibiotics	> 1000 PMN	↓	↑	↑	Turbid / Cloudy
TBM AIDS + PEM gdtv + wks	> 100-1000 lymphocytes	↓	↑↑ Cob web Coagulum	↑	Clear / Straw
Viral meningitis ↕ encephalitis days	> 25-100 lymphocytes	normal	> 1g/dL ↑	↑	Clear

Q. 7-year-old child has been sick for 4 weeks with persistent fever, irritability, vomiting, and recent onset of headache and neck stiffness. For past one day mother says child is in altered sensorium and not recognising the mother. Lumbar puncture reveals: opening pressure raised, protein 180 mg/dL, glucose 35 mg/dL (blood glucose 100 mg/dL), and lymphocytic predominance (120 cells/mm<sup>3</sup>). What is the most likely diagnosis?



(n) CSF glucose  
 $> 0.6$

$$\frac{35}{100} = 0.35$$

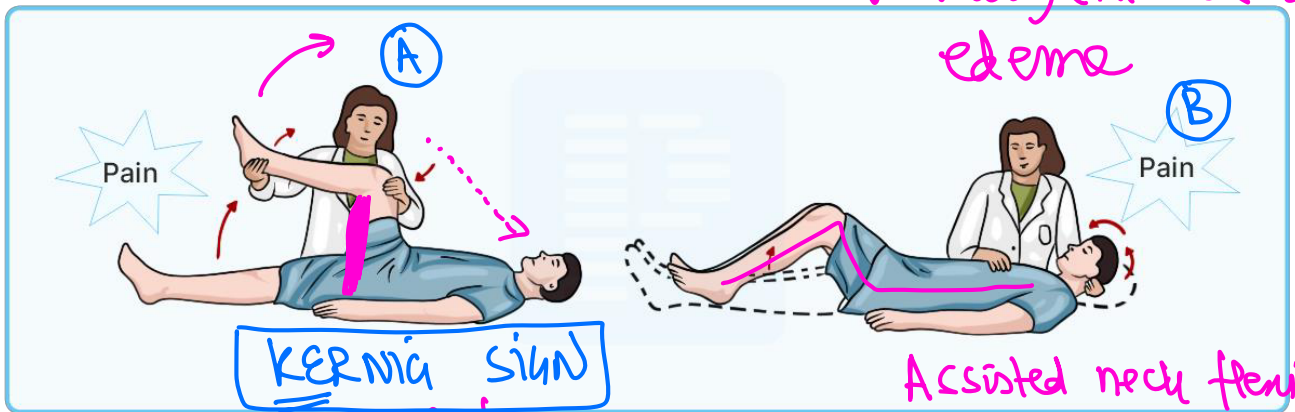
- a. Viral meningitis
- b. Pyogenic meningitis
- c. Tuberculous meningitis
- d. Cryptococcal meningitis

IOC  $\Rightarrow$  LP: CSF CBNAAT for MTB  
 CSF CULTURE

Rx: A.T.T x 1 year

+ DEXAMETHASONE: 6 wks :  $\downarrow$  vasogenic cerebral edema

L (liquid): MIDDLE BROOKS media



**KERNIG SIGN**

Assisted neck flexion

EXTENSION of knee  
 stretch HAMSTRING muscle: spasm  
 pt. wince  $\bar{c}$  pain

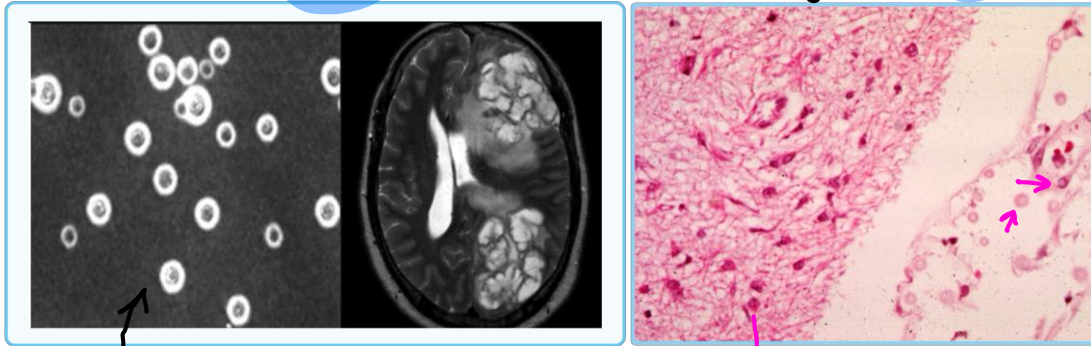
unvoluntary HJ flexion  
 KJ

**BRUDZINKI SIGN**

Signs of Meningeal irritation  $\checkmark$

Q. HIV positive truck driver comes with fever and headache for the past 3 days. He also has photophobia and cannot drive. On examination nuchal rigidity is noted. Fundus shows papilledema. CSF preparation and MRI head is shown. Diagnosis is?

IOC: CSF LATERAL Flow assay > urine lateral flow assay



INDIA ink stain

Mycetozome

**Mode of entry of fungus into patient**

**Work up**

1. MRI head → Soap bubble app, Raised ICP
  2. Guarded Lumbar puncture and CSF cytology → L++  
Sugar: ↓  
PROTEIN: ↑
  3. Urine or blood lateral flow assay for antigenic detection for cryptococcus
- Treatment = Liposomal Amphotericin B + 5-flucytosine iv 2wks ← initial

\* oral fluconazole: till 12 months

2° prophylaxis of cryptococcus  
 CD4 count: > 100 cells/cu.mm  
 PCR HIV RNA < 500 copies/mm

Route of acquiring infection

1. Pigeon droppings inhalation
2. Eucalyptus TREE BARK

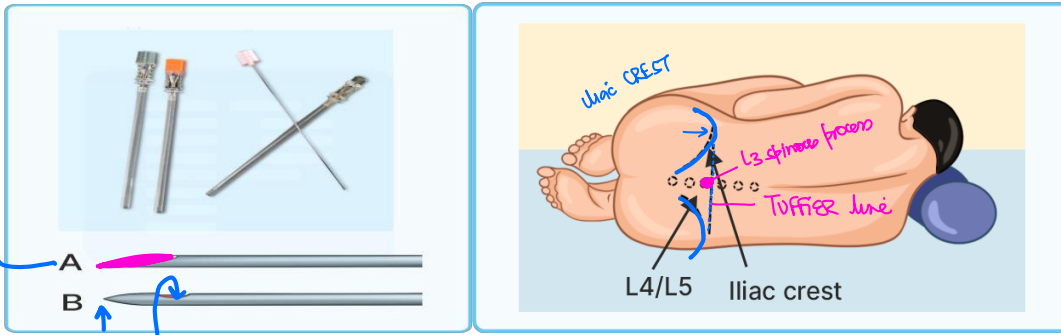
CD4: < 500	TB, KS	CEREBRAL
< 200	P. JIROVECI pneumonia	Toxoplasmosis
< 100	CRYPTOCOCCUS Neofarmanis,	<u>CRIBLES</u>
< 50	CMV Retinitis	

# Lumbar puncture

L3-L4

Preferred site

QUINCRE



A  
B  
C: WHITACRE SPROTTE

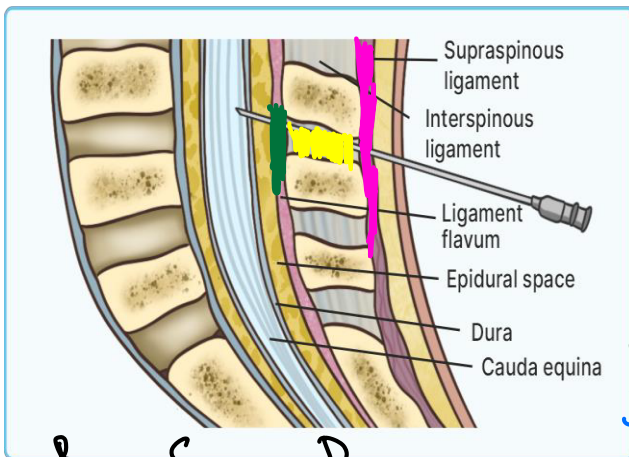
## Contraindications to LP

1. Raised ICP
2. Local site infection PYODERMA
3. Kyphoscoliosis

\* 4. bleeding diathesis INR > 1.7

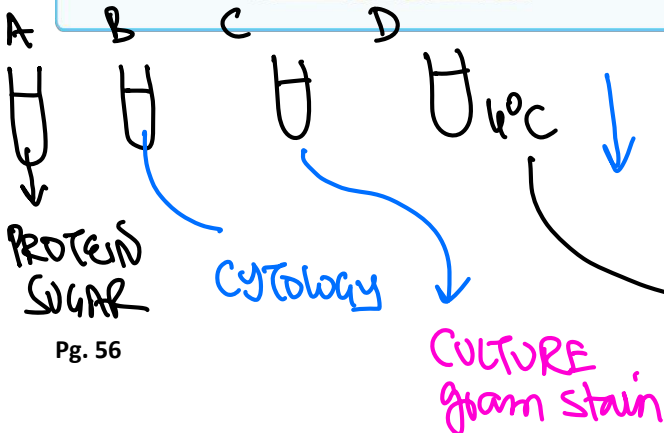
- Highest point of iliac crest joined to form Tuffier line that cuts through <sup>L3</sup> spinous process

- Most common complication of LP is headache. Best drug for management of PDPH = IV caffeine > opioids



## LAYERS PIERCED during LP

1. Skin sc
2. SUPRASPINOUS ligament
3. INTRASPINOUS =
4. lig. flavum
5. DURA
6. Arachnoid matter CSF

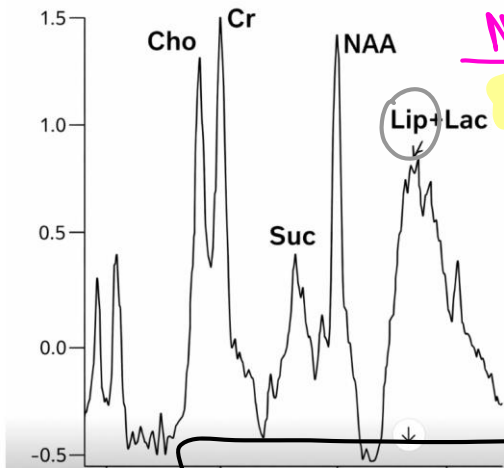
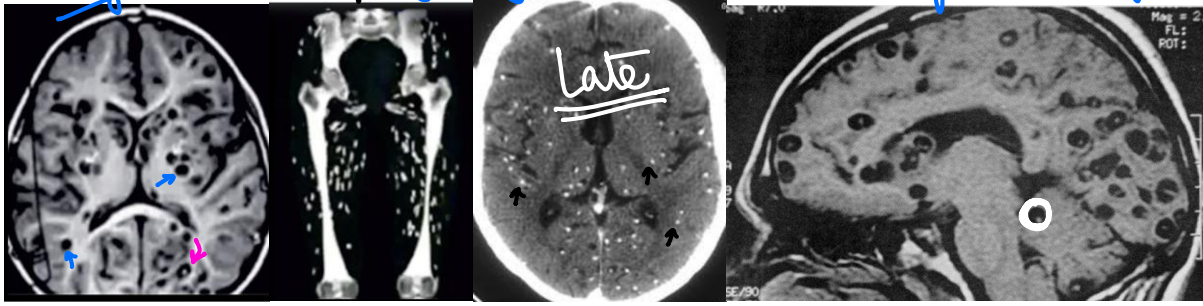


RICE grain calcification: cryptococcosis

Neurocysticercosis (caused by ingestion of proglottids containing eggs of tenia solium)

Early

fecally contaminated soil grown vegetables \*



NCC vs TUBERCULOMA

MRS

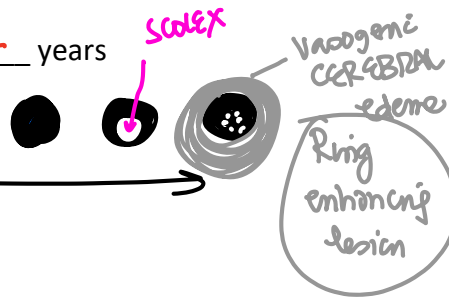
lipid PEAK = TB  
amino Acid = NCC

epileptogenic foci

Rx: Calcified lesions: Only **Valproate x 2** years

Rx: Hypointense lesions with/without scolex

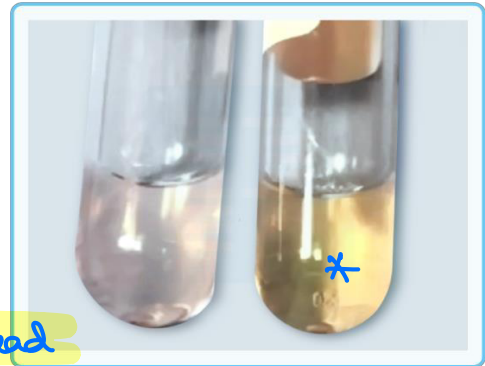
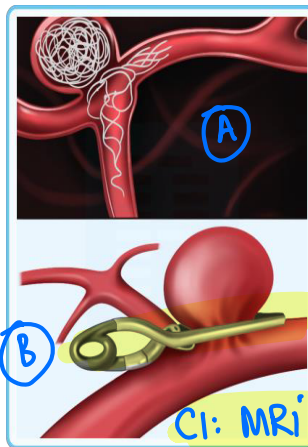
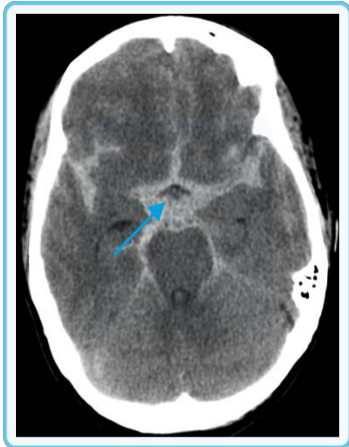
1. DEXA METHASONE IV
2. Albendazole
3. Valproate x 2 years



DIFFERENCE BETWEEN NEUROCYSTICERCOSIS AND CNS TUBERCULOMAS LESIONS

NEUROCYSTICERCOSIS	TUBERCULOMA
1. Lesions are <20 mm and may be single or multiple.	1. Often multiple and >20 mm because of conglomeration.
2. Meningitis feature is not there.	2. Meningitis is usually associated.
3. Present at grey-white matter junction.	3. More common in posterior fossa.
4. Other involvements like eyes, muscles or subcutaneous tissues.	4. Spread is mostly secondary to infection somewhere else.
5. T2W shows hyper intensity with hypointense scolex in it. No midline shift and ring enhancement is there depending upon the staging.	5. Hy pointensity seen in T2W and midline shift may be present.
6. MR spectroscopy shows multiple amino acid peaks.	6. MR Spectroscopy shows lipid peak.

## Subarachnoid hemorrhage



CI: MRI head

### Buzz Words

- Worst headache of my life + nuchal rigidity
- Thunderclap headache (also seen in Pituitary apoplexy, CVT, Hydrocephalus acute, HTN crisis): occurs without warning and reaches peak intensity in 1 minutes

-Sudden onset Headache with

✓ -Next best investigation after CT head →

SAH evidence CEREBRAL ANGIOGRAPHY

✓ -Next best investigation if CT head is normal →

L.P guarded: RBC in CSF

- Leading cause of SAH: Trauma > rupture of berry aneurysm

• First investigation

Headache (hours): NCCT head  
nuchal rigidity +, brudzinku sign ⊕

Xanthochromic CSF

blood in syring F  
Star of death

• Next best investigation after NCCT head

↳ CEREBRAL angiography

• What to do if CT head is normal → guarded L.P

Tint lemon yellow

RBC ++ → Xanthochromic

• Finding of guarded Lumbar puncture

• Treatment of choice: Endovascular coiling > Aneurysmal Clipping

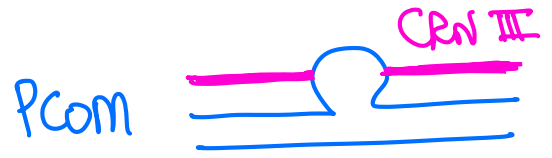
# Nimodipine : 1<sup>o</sup> protective vasospasm

- DOC of choice

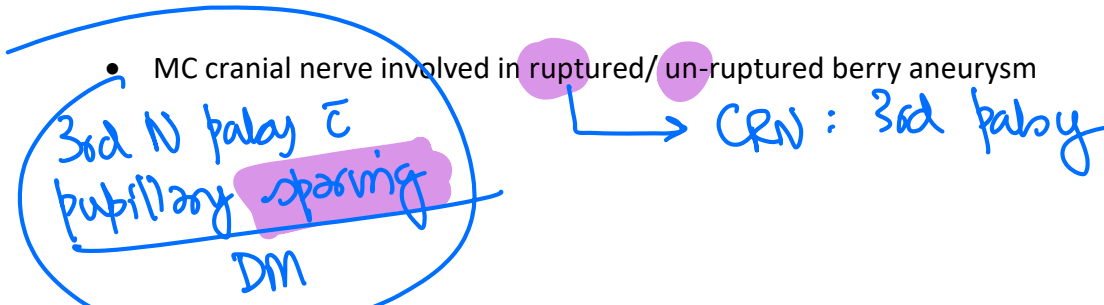
- 1st step in management of SAH : 1. Nimodipine  
 NCCT head evidence 2. CEREBRAL anjio  
 3 Endovascular coiling

- MC location of Berry aneurysm ACOM

- MC location of Berry aneurysm prone to rupture



- MC cranial nerve involved in ruptured/ un-ruptured berry aneurysm



## Leading cause of death in SAH

1. Delayed cerebral ischemia — VASOSPASM
2. Hydrocephalus
3. Bleeding
4. Rebleeding

- \* 5. Electrolyte imbalance

BNP +++ : NATRIURESIS :  $Na^+ < 125$   
 SEIZURES +

CEREBRAL SALT WASTING SYNDROME

Hypovolemic  
Hyponatremic

Urine sodium: ↑  
 S. sodium: ↓

Euvolemic : SIADH

\* EDH

← acute SDH

Q. Diabetic patient falls in washroom and head slams against the wall. Lucid interval was noted. From the next day onwards, her son notices she is becoming irritable and sleeps for most part of day. He also tells that she is having severe headache and has trouble speaking and recollecting events. NCCT was done on day 3 and is shown below?



Rt sided. acute subdural hemorrhage  
↑  
HYPERDENSITY  
Concave-Convex  
Rx  
Mannitol / Burr hole / Craniotomy

cal evacuation if they do not enlarge. Stupor or coma, hemiparesis, and unilateral pupillary enlargement are signs of larger hematomas. The bleeding that causes larger subdural hematomas is primarily venous in origin, although arterial bleeding sites are sometimes found at operation, and a few large hematomas have a purely arterial origin. In an acutely deteriorating patient, an emergency craniotomy is required. In contrast to epidural hematomas, there is significant morbidity and mortality associated with acute subdural hematomas that require surgery.

\*

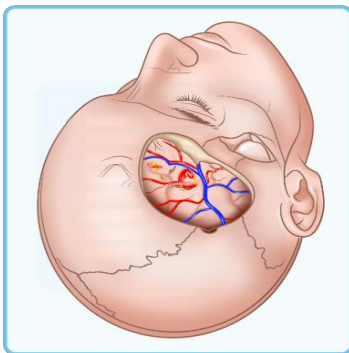
decompressive  
Hemicraniectomy  
↳ malignant cerebral edema  
\* Hemorrhagic stroke



Q. Diabetic patient falls in washroom and head slams against the wall. Over the next 3 weeks she becomes forgetful and delirious. NCCT was done.



\* Rt chronic subdural hemorrhage  
↓  
Hypodensity  
Rx: Burr hole St



acute and chronic hematomas.

Clinical observation coupled with serial imaging is a reasonable approach to patients with few symptoms and small chronic subdural collections that do not cause mass effect. Treatment with surgical evacuation through burr holes is usually successful, if a cranial drain is used postoperatively. The fibrous membranes that grow from the dura and encapsulate the collection may require removal with a craniotomy to prevent recurrent fluid accumulation.

